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Зміст

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Infant Colic

This is a 20 day old newborn that is brought to the emergency department at 10 pm with a chief complaint of extreme fussiness. His parents think he has abdominal pain as he is "gassy" and pulls his legs up as if he is trying to stool. He passes a lot of gas from his rectum and his parents can hear his stomach gurgling a lot. Tonight’s episode has lasted for 4 hours with intractable crying, and his parents are very distraught. They have tried feeding, a pacifier, rocking, burping, changing the diaper, and inserting a rectal suppository but nothing has relieved the crying. He is currently feeding a standard cow's milk formula with iron without vomiting or diarrhea. Further questioning reveals this is the fourth day in a row that this has happened on a daily basis, usually in the evening, but the baby usually cries for about 2 to 3 hours.

He was born at term with no prenatal problems or infection at time of birth. No maternal use of illegal drugs. He has been feeding well with good weight gain and no fussiness until 4 days ago (age 16 days of age). No apnea, no vomiting, no fever, no constipation, no seizure activity, no trauma or history of shaking or abuse. He has been acting normally between daily episodes of fussiness.

Exam: VS T 37.0, P 130, RR 32, BP 80/55, oxygen saturation 100% in room air. Height, weight and head circumference are at the 50th percentile. He is a healthy appearing infant who is not crying at this time. He is alert and active. HEENT: Soft fontanelle, good eye contact. No evidence of corneal abrasion or watery eyes. Vigorously feeding during exam. No signs of closed head injury. Neck, heart and lung exams are normal. His abdomen is soft and non-distended. There is no definite tenderness. Bowel sounds are active. He has no inguinal hernias. His testes are normal. No tourniquets are noted over his penis and digits. He is moving all extremities well and his muscle tone is normal. He has no pain on movement. Color and perfusion good. No pallor or mottling of his skin is present.

Diagnostic impression by the physician: Unexplained recurrent crying with normal physical examination. Unclear etiology.

Colic is one of the most commonly made diagnoses during the first 4 months of life with a reported incidence of 10% to 35% of all infants. The word "colic" is derived from the Greek word "kolikos", which refers to the large intestine. Colic has also been called the three month colic, infant colic syndrome, or paroxysmal fussing in infants. The classic definition of infantile colic was described by Wessel in 1954 as, crying lasting more than 3 hours per day, 3 days per week, and continuing more than 3 weeks in infants less than 3 months of age. During these paroxysms, the legs are often flexed, the infant may be described as gassy, and parents often think the infant has abdominal pain. In addition, crying is not relieved by normal parental interventions (feeding, burping, changing diapers, etc.).

How much crying is normal? In 1962, Brazelton published characteristics of the median daily crying at various ages: At 2 weeks of age: 1 hour and 45 minutes. At 6 weeks of age: 2 hours and 45 minutes. At 12 weeks of age: less than 1 hour. The peak time for crying is 3:00 pm through 11:00 pm ("prime time"). Infants whose crying significantly exceeds these median values could be labeled as having "colic"; however, this is also dependent on the parents' ability to cope with crying and as to whether they label their infant's behavior as "normal crying" or "abnormal crying" (i.e., colic).
The four clinical signs of colic are: 1) paroxysmal onset, 2) distinctive high-pitched pain cry, 3) physical signs of hypertonia and 4) inconsolability. Colic presents as intermittent and unexplained crying during the first three months of life by babies that are otherwise healthy. The "infant colic syndrome" (paroxysmal fussing) basically involves cyclic discrete periods of intractable crying, usually on a daily basis, with onset at 1-4 weeks of age (may be as early as the first week of age) and dramatic spontaneous improvement by 3-4 months of age. In addition to infant irritability, colic is characterized by recurrent episodes, excessive restlessness or activity, or diminished consolability. Colic is distinguished in that the crying is paroxysmal, intense and different in type from normal fussing and crying.

The defining elements of colic, according to Carey are: full force crying for at least 3 hours per day, for 4 or more days per week, in infants who are less than 4 months old and are otherwise healthy. The infant begins a colic episode with a paroxysmal or sudden onset of crying. The cry reaches a screaming level, is often high pitched and coupled with facial grimacing indicating that the infant is in severe pain. There is increased motor activity, which may include flexion of the elbows, clenched fists, and generalized hypertonicity of the musculature, with the knees drawn up or legs stiff and extended. Milder cases of "colic" may exist, but defining this would be difficult.

There is no clear understanding of the etiology, pathophysiology and treatment of colic; however, proposed models for the etiology of colic fall into 3 broad categories: intrinsic or biological factors in the infant, extrinsic factors in the psychosocial environment and an interaction or systems approach.

The most important thing to remember about infants who present with intractable crying is this: ALL THAT CRIES IS NOT COLIC! Crying is a non-specific response in an infant, which may be a major symptom of an underlying pathologic process. The etiologies of intractable crying in infancy range from a benign phase of psychomotor development to a life threatening illness. The etiology is initially obscure and an accurate diagnosis is dependent on a knowledgeable and organized approach. A careful history and physical exam with selected laboratory studies usually establishes a diagnosis.

Since most of these patients initially present to the emergency department, the emphasis is on the evaluation of the infant or young child with intractable crying, and one must exclude serious underlying illness. In Poole's 1991 study in afebrile infants, those who ceased crying before or during the initial assessment were unlikely to have a serious underlying illness, whereas the persistence of excessive crying after the initial examination was predictive of a serious underlying process. Therefore, do NOT discharge an infant or young child with persistent, excessive crying. Look for "red flags" in the history and physical, which suggest the possibility of significant underlying pathology (see Tables 1 and 2). The presence of any of these "red flags" should prompt a more extensive evaluation and aggressive management, often including specialty consultation and hospitalization (e.g., meningitis or sepsis).

Robert Bolte has described "Red Flags" of non-colic causes of extreme fussiness, which may be signs or symptoms of life threatening illness, obtained by further history or physical examination. ANY OF THESE RED FLAGS SUGGEST NON-COLIC ETIOLOGIES OF FUSSINESS and must lead to extensive evaluation and aggressive management (Tables 1 and 2). Do not make a diagnosis of colic on patients with any of these historical or physical examination "red flags" until other causes listed under "differential diagnosis" (Table 3) are ruled out.

1. **Table 1 - Historical "Red Flags" Associated with Intractable Crying in Infancy**
   1. Fever (>38 degrees C, 100.4 degrees F, rectal) in an infant less than twelve weeks of age.
   2. Paradoxical irritability (infant doesn't want to be held).
   3. Premature rupture of membranes (>24 hours), perinatal maternal fever/infection, neonatal jaundice.
   5. Poor feeding, poor weight gain.
   6. Significant decrease in level of activity, cyanotic/apneic "spell", or seizure-like episode.
   7. Bilious or projectile vomiting.
   8. History not suggestive of classical "infant colic syndrome".
   9. History suggestive of physical abuse (injury not consistent with reported history, inappropriate delay, non-maternal caretaker).
   10. Antibiotic pre-treatment ("partially treated" sepsis/meningitis), particularly in the young infant.
   11. History of recent head trauma.

2. **Table 2 - Physical Examination "Red Flags" Associated with Intractable Crying in Infancy**
   1. Fever (>38 degrees C, 100.4 degrees F, rectal) in an infant less than twelve weeks of age.
   2. Hypothermia.
   3. Heart rate >230.
   4. Lethargy, poor eye contact.
Table 3 - Differential diagnosis of Infant Colic Syndrome:

I. Infectious

1. 1) otitis media
2. 2) meningitis/sepsis
3. 3) encephalitis
4. 4) urinary tract infection
5. 5) osteomyelitis, septic arthritis
6. 6) pneumonia
7. 7) gingivostomatitis, pharyngitis
8. 8) gastroenteritis
9. 9) Kawasaki Disease

II. Trauma

1. 1) child abuse - shaken baby
2. 2) corneal abrasion or foreign body in eye
3. 3) accidental fracture/musculoskeletal injury

III. Gastrointestinal/Genital

1. 1) intussusception
2. 2) reflux esophagitis (GERD)
3. 3) constipation/anal fissure
4. 4) midgut volvulus
5. 5) incarcerated inguinal hernia
6. 6) appendicitis
7. 7) milk protein intolerance
8. 8) testicular torsion
9. 9) penile tourniquet (from hair)

IV. Nutritional

1) underfeeding

V. Respiratory
1) hypoxemia/hypercapnia

VI. Metabolic

1. 1) hyponatremia, hypernatremia
2. 2) metabolic acidosis
3. 3) hypocalcemia/hypercalcemia, hypoglycemia, hyperglycemia
4. 4) inborn errors of metabolism

VII. Integument

1. 1) diaper dermatitis
2. 2) atopic eczema
3. 3) burns (accidental and non-accidental)
4. 4) foreign body (pin)
5. 5) hair encirclement (strangulation of digit, penis, clitoris, uvula) diagnosed by a thorough physical exam
6. 6) bites and stings
VIII. Drugs and Toxins

1. 1) neonatal narcotic withdrawal
2. 2) neonatal barbiturate, ethanol, hydantoin withdrawal
3. 3) irritability related to smoking mothers who breastfeed
4. 4) reaction to pertussis immunization
5. 5) theophylline, antihistamine, decongestant, cyclic antidepressant, amphetamine, cocaine toxicity

6. A thorough history and a meticulous physical exam are the cornerstones of accurate diagnosis. Poole described 56 afebrile infants who presented with unexplained excessive crying to the emergency department. The history provided clues to the final diagnosis in 20% of the cases, while the physical exam revealed the final diagnosis in 41% of the cases and provided clues to the final diagnosis in another 11%. Physical examination must start with a 2 or 3 minute period of observation from a distance with the child undressed, on the parent's lap. Assess the patient's appearance, distractibility, alertness, eye contact, ability to be comforted, respiratory rate and pattern, spontaneous extremity movement, etc. The extent of your work-up is usually determined from this observation period. Special emphasis should be given to the examination of the skin, palpation of the abdomen, eye examination (with funduscopic and eversion of the eyelids), evaluation of anterior fontanelle fullness, inspection of the tympanic membranes, oropharynx, and gums, palpation of extremities and clavicles, and performance of an anal rectal exam which may be done with a cotton tip swab.

If colic is determined to be the likely diagnosis, there have been a number of studies with varying results regarding treatment:

1. 1. Taubman's behavior-modification approach provides useful information for counseling parents (Table 4). His behavior-modification approach resulted in a 65-70% decrease of crying time (3.2 to 1.1 hours per day) in colicky infants in his 1984 and 1988 studies and a similar reduction in the crying time (3.8 to 1.1 hours per day) in a 1998 controlled study by Dihigo. This "good" approach assumes that colic results from inadvertent failure to respond to the infant's desires. The infant's crying is not a "cry of pain" but rather a way to communicate a need or desire. Taubman also described a "bad" approach (ignoring the baby) which assumes colic that results from over stimulation, therefore generally "ignoring" the baby (letting them cry) would be the logical treatment. The ignoring approach did not result in any decrease in the crying time in Taubman's 1984 study.
2. 2. Simethicone (Mylicon, OTC) (10) (a non-toxic "defoaming" agent). The apparent effectiveness of simethicone (seen within 1-4 days in 54-67% of treated infants) probably represents a high-grade placebo effect. Simethicone converts gas foam into non-foam gas, but the gas remains in the bowel lumen.
3. 3. Herbal tea (commercially available chamomile tea). Weizman, et al showed that 57% of colicky babies improved (vs. 26% placebo), 5 oz. tea per dose with each colic episode not to exceed three times per day.
4. 4. General counseling . Empathy and describing the natural history of colic to parents results in improvement by 3-4 months. Increased carrying time, automatic rocker swings, driving around the neighborhood (with baby in a car seat) and nap-time swaddling are benign measures that may be helpful.

Paregoric (tincture of opium), Bentyl (dicyclomine, possible association with SIDS) and Levsin (hyoscyamine sulfate, associated with anticholinergic toxicity) should NOT be used. Placing the infant in a car seat on the washing machine should NOT be used because of the possibility of falls and secondary head injury. Empiric formula changes are generally not useful, but this is a benign measure and it is often suggested. Mothers who are breast and bottle feeding should be encouraged to breast feed as much as possible and minimize formula feeding. Infants who are exclusively formula fed can be changed to a protein hydrolysate formula (Nutramigen, Pregestimil, Alimentum), as a trial to see if there is a beneficial response.

1. 1. Table 4 - Good Colic Advice" for Parents: The underlying assumption of this advice is that continued crying in colicky infants results from the parents' inadvertent failure to respond to their infant's desires which the cries are signaling to the parents. The infant's crying is not a "cry of pain" but rather a way to communicate a need or desire.
2. 2. Try to never let your baby cry.
3. 3. In attempting to discover why your infant is crying consider these possibilities:
   a. The baby is hungry and wants to be fed.
b. The baby wants to suck, although he/she is not hungry.
c. The baby wants to be held.
d. The baby is bored and wants stimulation.
e. The baby is tired and wants to sleep.
f. The baby needs his/her diaper changed.

1. 3. If the crying continues for more than 5 minutes with one response, then try another.
2. 4. Decide on your own in what order to explore the above possibilities.
3. 5. Don't be too concerned about overfeeding your baby.
4. 6. Don't be too concerned about spoiling your baby.

When infant crying continues despite all efforts to stop it, including feeding, do the following:

1. 1. Put the baby in the crib and let him cry for up to one-half hour.
2. 2. If still crying, pick the baby up for a minute or so to calm him/her then return him/her to the crib.
3. 3. Repeat the above until the infant falls asleep or three hours have passed.
4. 4. After three hours, feed the baby.
5. 5. Do not shake the baby.

In summary, it is important to remember that crying is a non-specific response in an infant, which may be a major symptom of an underlying severe pathologic process and NOT necessarily just "colic." A careful history and examination combined with selected laboratory studies usually establishes a diagnosis. Crying may simply be a normal response to stress such as hunger, discomfort, or over or under-stimulation, or may represent the "infant colic syndrome" (paradoxical fussiness). Close follow-up is crucial if the etiology of the irritability and excessive crying is still somewhat obscure at discharge. Do not discharge an irritable infant if "extreme fussiness" has not resolved, particularly if a "red flag" is present.

Questions
1. Which of these are NOT a feature of the infant colic syndrome?
   a. distinctive high-pitched pain cry
   b. inconsolability
   c. paroxysmal onset
   d. vomiting
2. Which of these is correct?
   a. colic usually occurs in infants greater than 3 months of age
   b. fever often accompanies colic
   c. colic is very rarely seen
   d. none of the above are correct
3. All of the following are correct regarding historical red flags, except:
   a. Red flags suggest that this intractable crying infant may not be due to the classic "infantile colic syndrome".
   b. Red flags include head trauma.
   c. Red flags exclude maternal illicit drug use.
   d. Red flags include paradoxical irritability.
4. Physical red flags include which of the following (check all that apply):
   a. fever
   b. lethargy
   c. poor feeding
   d. abdominal tenderness
5. True/False: Good advice for parents assumes their infant is trying to communicate a need or desire resulting from the parents inadvertent failure to respond to their infant's desires.
6. An acceptable approach(es) to infant colic include(s):
   a. Let the baby cry and ignore the baby.
   b. Put the baby in a car seat on the washing machine.
   c. Shake the baby to sleep.
   d. Try to discover why your infant is crying.

Answers to questions
1. d, 2.d, 3.c, 4.abcd, 5.true, 6.d
Abdominal Pain

This is a 6 year old female presenting with a 2 day history of crampy abdominal pain. The pain is located in the upper mid-abdomen and is associated with anorexia, nausea and four episodes of green vomitus. She appears to be weak and her parents noticed a decrease in urination. There is no history of diarrhea, trauma, fever or coughing. She has not passed any stools for the two days that she has been ill.

Her past history is significant for an appendectomy one year ago. Her family history is negative for other family members with similar problems.

Exam: VS T 37.0, P110, R 12, BP 100/60. She is alert and subdued. She moves without difficulty but cries episodically because of crampy pain. Her mucous membrane are sticky. Her eyes are sunken. Her neck is supple. Her heart and lungs are normal. She has a RLQ (McBurney's point) scar. She has moderate abdominal distention with hyperactive bowel sounds, peristaltic rushes and borborygmi with generalized mild tenderness. She has no inguinal hernias and her external genitalia are normal. A rectal exam finds no stool or mass. Her back is non-tender. Her skin turgor is decreased, but her overall color and perfusion are good.

CBC: WBC 14.0, Hgb 16, Hct 48, K 3.0, Cl 90, bicarb 30. Urinalysis: SG 1.030, no pyuria or hematuria. Abdominal series radiographs show distended ladderlike small bowel with large air/fluid levels and no large bowel gas. No calcifications. Lung bases are normal.

Impression: Small bowel obstruction secondary to adhesions; dehydration, metabolic alkalosis and hypovolemia.

Abdominal pain is a common symptom of childhood. Its importance lies in differentiating the vast majority self-limited causes of pain from those few conditions that may be life threatening. In the latter category are those conditions that lead to a diagnosis of an “acute abdomen,” usually leading to surgical intervention. Examples of these in children are most commonly acute appendicitis followed by incarcerated inguinal hernias, bowel obstruction, traumatic injury, ovarian torsion, pancreatitis, and biliary disease. Further complicating the diagnosis is the young child’s relative inability to communicate and his/her inability to evaluate the abstract concept of pain.

In general, it is helpful to classify abdominal pain into two large categories: 1) pain originating in a hollow viscus, and 2) pain originating in a solid organ or the peritoneum.

Hollow viscus pain such as that of an the obstructed ureter, intestine, and gallbladder is colicky or spasmodic in nature. It coincides with the peristaltic waves of the organ as it attempts to overcome the distal obstruction such as ureteral or cystic duct stone or a fecal bolus in constipation. These waves or cramps are exactly what we experience with early acute appendicitis and gastroenteritis and are somewhat ameliorated by writhing and massage.

On the other hand peritoneal and solid organ pain such as caused by infection or trauma is aggravated by motion caused by coughing, abdominal compression, and walking. It is usually unrelenting or steady.

The search for the cause of abdominal pain is a good example of both inductive and deductive reasoning. In gathering data, a complete history and physical examination should suggest a disease process, a hypothesis or diagnosis (induction) which in turn should suggest a search for confirmative or corroborative data to strengthen or disprove the diagnostic hypothesis (deduction).

In evaluating the case above using inductive reasoning, the symptoms of crampy mid-abdominal pain, bilious vomiting, and history of prior abdominal surgery, suggest a hypotheses of bowel obstruction. If it is intestinal obstruction, an abdominal series should show an obstructive pattern (deduction).

It could be ureteral colic but this is uncommon in children and there is no blood in the urine indicating that a ureteral stone is unlikely. It could be biliary colic but this is rare in children and the pain distribution is not that of biliary pain. It could be a gynecological problem but this girl is prepubertal, and ovarian torsion frequently presents in the lower quadrant and radiates to the anterior thigh.

With a bowel obstruction, there may be bowel infarction. If there is gangrene by deduction there should be an elevated WBC, absent bowel sounds, marked tenderness, and localization of pain. Since none of these findings is present, bowel compromise (infarction) is unlikely.

The following data suggest dehydration by induction: urine specific gravity of 1.030, history of infrequent urination, sticky (dry) mucous membranes, sunken eyes and weakness.
In addition to peritoneal and hollow viscus pain, there is pain of neural origin. Nerve root compression by spinal cord tumors are rare but must be suspected if no other cause for the discomfort can be found and if the pain distribution is that of a dermatome. There should be no tenderness to palpation, but there may be hypesthesia.

Inflammation of the pleura from a pneumonic process in the distribution of the lower thoracic nerves is not an infrequent cause for referred abdominal pain and should be a reason for auscultation of the chest in a search for pneumonia or pleurisy. The abdominal series includes the lung bases and should be noticed when evaluating abdominal films. Lower lobe pneumonia can frequently be seen in the lung portions of an abdominal series, and it is very frequently overlooked since the clinician is usually focusing on the abdominal structures.

Diabetic acidosis, lupus erythematosus, porphyria, and other systemic illnesses may cause pain and inflammation of the serous surfaces (serositis). Some non-surgical causes of abdominal pain are lactose intolerance, inflammatory bowel disease, intussusception (sometimes requires surgery), Henoch-Schonleinpurpura, ascariasis and acute gastroenteritis. Of help in the diagnosis of many of the non-surgical diseases is their chronicity or recurrence. Of course, the first occurrence of the symptoms is always more difficult to sort out.

Constipation is a common cause of chronic, recurrent and acute abdominal pain of varying degrees of severity. Relief after an enema is characteristic, but some cases are associated with more serious GI pathology, since the presence of constipation does not rule out the presence of something else, such as appendicitis.

As with most rules of thumb or generalizations there are exceptions that the clinician should keep in mind. One of these is that appendiceal pain always occurs in the right lower quadrant since the appendix is located there. However, since it is 6-13 cm long, its inflamed tip may come to rest anywhere in a radius of 6-13 cm from its base. This means that tenderness may be produced in the right upper quadrant, the midline, or in the suprapubic region. Similarly, if it is retrocecal so that it has no contact with peritoneum, the child may not exhibit severe tenderness. In its retrocecal position however, it may rest on the right psoas muscle and cause pain with active right hip flexion. If it lies on the right ureter, hematuria and pyuria may be produced.

Malrotation of the cecum may lead to all sorts of additional presentations for acute appendicitis. However, the astute clinician should keep in mind that rare things occur rarely and that when you hear hoof beats they are most likely horses and not gazelles or camels.

The examiner of children must realize that most children wish to please, so that a child brought in the middle of the night to the hospital may feel obligated (obliged) to its adult caregivers and nighttime physicians to show cause for such concern. Thus, when asked if their tummy hurts, they may be inclined to answer affirmatively to justify the trip and trouble.

Similarly, older teenage boys with a macho image to uphold, may hesitate to admit pain and/or tenderness. It is a useful ploy to engage the child/teen in conversation about his or her dog, siblings or other familiar childhood topics while depressing the abdominal wall. Any true tenderness will be confirmed or refuted by involuntary guarding or its absence. A useful technique is to ask the child to cough while asking what he or she feels. This ploy will direct attention away from the abdomen but almost always elicits peritoneal discomfort if present.

Persistence and constancy of a sign heightens its importance in diagnosis. Tenderness should be reproducible. Sensory innervation of the intestines is via the ninth through eleventh thoracic nerve roots. Consequently pain from the intestines due to stretching is appreciated as originating from the mid-abdomen until an inflammatory process localizes it in the dermatome of the parietal peritoneum. There are several areas of referred pain which, when present, may suggest a specific entity. Radiation of flank pain into the groin and ipsilateral scrotum or labium suggests ureteral colic. Lower quadrant pain radiating to the anterior thigh should suggest torsion of the ipsilateral ovary and tube. A point of pain in either shoulder indicates subdiaphragmatic irritation from blood or pus. Right upper abdominal pain radiating around to the back suggests biliary tract involvement but epigastric pain radiating through to the back suggests a pancreatic origin.

The use of specific diagnostic tests should be guided by the clinical examination and evaluation. They should not be a substitute for such evaluation and should not precede the clinical examination since the clinical appraisal may obviate the need for additional tests. Plain film radiographs, ultrasound, computerized axial tomography (CAT scan), magnetic resonance imaging (MRI), and contrast studies may aid in the evaluation of abdominal pain but should be used judiciously. The flat and upright plain film radiographs can be particularly useful in recognizing small bowel obstruction, ileus, abnormal calcifications and lower lung pathology.

In summary, acute abdominal pain is a common childhood complaint. In most instances it usually passes without much interruption of the events of daily living. However, abdominal pain can also signal severe illness leading to serious morbidity and death if not attended to. Thus, separating the chaff from the wheat is extremely important. Persistence of pain associated with vomiting, dehydration and signs of inflammation should not be ignored, but should stimulate a thorough evaluation. The use of both inductive reasoning to formulate a hypothesis for the cause of the pain followed by deductive reasoning...
to confirm the hypothesis is the basis for identifying the correct diagnosis.

Questions

1. 1. True/False: Surgical causes of abdominal pain are much less common than non-surgical causes.
2. 2. True/False: Predicting a finding from a hypothesis is called deductive reasoning.
3. 3. What characteristics differentiate hollow viscus from solid viscus and peritoneal pain?
4. 4. Pain from distended intestines is appreciated in what area?
5. 5. Where is the pain of urogenital origin referred?

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Answers to questions

1. True
2. True
3. Crampy (hollow viscus) versus steady (solid viscus and peritoneal).
4. Mid-abdomen
5. Flank, groin and ipsilateral scrotum or labium

Gastroenteritis and Dehydration

An 18 month old male is brought to the emergency department with a chief complaint of diarrhea and vomiting for 2 days. His mother describes stools as liquid and foul smelling, with no mucous, slime or blood. He reportedly is unable to keep anything down, vomiting after every feeding, even water. He has about 6 episodes of diarrhea and 4 episodes of vomiting per day. His mother reports that he is not feeding well and his activity level is decreased. He seems weak and tired. He has a decreased number of wet diapers. He attends daycare during the day when he is well. His last weight at his 15 month check was 25 pounds (11.4 kg).

Exam: VS T 37.0, P 110, RR 25, BP 100/75, weight 11.3 kg (40th percentile). He is alert, in mother's arms, crying at times, and looks tired. HEENT: anterior fontanel closed, minimal tears, lips dry, mucous membranes tacky, no oral lesions or erythema, TMs normal. His neck is supple. Heart exam reveals mild tachycardia, no murmur. Lungs are clear. His abdomen is flat, soft, and non-tender with hyperactive bowel sounds. Testes are descended, non-swollen, non-tender. His diaper is dry. He moves all his extremities. No rashes are present. His capillary refill time is less than 3 seconds and his skin turgor is slightly diminished.

He is given 40 cc/kg of IV normal saline over two hours in the emergency department, but when given 30 cc of fluid to drink in the ED, he is unable to hold this down and he passes another large diarrheal stool. He is then hospitalized for further management.

Acute gastroenteritis is an ailment that is very common among children. During the first 3 years of life, a child will likely experience about 1 to 3 acute diarrheal illnesses. Diarrhea is defined as an increase in fluidity and volume of feces. Nearly all diarrheal infections are transmitted via the fecal-oral route. Many bacterial etiologies are also food borne.

When evaluating a child with diarrhea and/or vomiting, several important points and observations in the history and physical can help to assess severity and determine its etiology and pathogen involved. Information on the number, volume, and/or fluidity of stools and emesis should be obtained. However, this can be rather cumbersome if the number of episodes is large since recording the volume of each stool and emesis is unrealistic and not very helpful clinically, once the number of episodes exceeds 5 to 10. A history of fever, blood or mucus in the diarrhea, foul odor to the diarrhea, a large quantity of diarrhea or diarrhea for a prolonged duration are suggestive of a bacterial etiology. Diarrheal "mucus" is
not something that is intuitive to most parents. The best question to ask is whether they have noticed any slimy, gooey, gelatinous, or mucous-like material in the diarrhea. The presence of mucus in the diarrhea is generally indicative of sheets of white blood cells in the diarrhea. The vomiting history should determine whether the vomitus is bilious or bloody (i.e., red or brown) and whether this is associated with abdominal pain. Parents will often convey that the vomitus contains mucus, but unlike the mucus in diarrhea, this is normal gastric mucus that is not helpful clinically. Other important historical items include: weight loss, dietary (intake) history, ill contacts and travel history. The physical exam should focus on signs of dehydration, conditions that may suggest an acute surgical condition and other systemic conditions which may cause these symptoms. The differential diagnosis of vomiting is extensive including systemic conditions such as meningitis, increased intracranial pressure, heart failure, pneumonia, urinary tract infection and many acute surgical conditions such as appendicitis, intussusception, midgut volvulus, etc. The differential diagnosis of diarrhea is more limited and is most often due to gastroenteritis.

Many organisms can cause acute infectious diarrhea and vomiting. These include bacteria, viruses, and parasites. Bacterial etiologies include: Campylobacter, E. coli, Staph aureus, Salmonella, Shigella, Vibrio, Yersinia and a few others. Viral gastroenteritis is by far more common. Amebic and parasitic etiologies are not very common in the United States, but such cases are treatable, making the identification of the pathogen essential.

Lab tests available include: CBC, stool Wright stain, stool culture, stool Rotazyme, serum electrolytes and glucose. In most instances, no laboratory studies are required. A CBC might raise the clinical suspicion of a bacterial etiology if a very high band count is present; however, this may also occur in viral etiologies, therefore it is not very helpful in most instances. A positive stool Wright stain identifies WBCs in the stool. This is suggestive of a bacterial etiology (similar to the history or observation of mucus in the stool), but since it is unable to determine the specific pathogen, it does not help clinicians in determining whether antibiotics are indicated. Thus, in most instances of gastroenteritis, a Wright stain is not helpful. A stool culture can be obtained by swabbing a diarrhea sample or by inserting a swab into the rectum, then rotating it to obtain organisms from the rectal mucosal surface. This latter method is called a rectal swab and it has a higher yield for identifying enteric pathogens, which are more likely to be found on the rectal mucosal surface than in the diarrhea itself. Thus, if a stool culture is to be obtained, it should be done via a rectal swab. A stool Rotazyme is a rapid test which identifies the presence of rotavirus in the diarrhea. A positive Rotazyme negates the need for a stool culture and antibiotic therapy. Serum electrolytes and glucose may be helpful in determining the degree of electrolyte imbalance, metabolic acidosis and hypoglycemia.

Viral gastroenteritis
Diarrhea and vomiting caused by viruses are usually self-limited. It usually manifests as watery diarrhea, without blood or mucus. The most common accompanying symptom is vomiting. Other symptoms include abdominal cramps, nausea, headaches, myalgias and fever. Four viral groups are regarded as medically important causes of acute gastroenteritis (rotaviruses, astroviruses, enteric adenoviruses, and calciviruses). They are all spread largely via the fecal-oral route.

Rotavirus accounts for 82,000 hospitalizations and 150 deaths per year in the United States. It is a very common cause of acute gastroenteritis in infants and children, responsible for over 50% of cases of acute diarrhea in children. Rotavirus causes severe illness in children 6-24 months. Most children (more than 90%) have been exposed to rotavirus by their 3rd birthday. It is the most common cause of diarrhea in infants and children in the winter months in colder climates, and is responsible for 35-50% of hospitalization for infants and children with acute diarrhea. Clinical features range from asymptomatic infection, to diarrhea preceded by severe vomiting. The incubation period is 2-4 days, and viral shedding occurs from a few days before, to 10 days after the onset of illness. newborns tend to have asymptomatic infections in the first few months of life, because transplacental antibodies and breastfeeding are protective. Clinical symptoms in infants and children usually consist of fever, abrupt onset of vomiting and watery diarrhea. One third of children will also have a concurrent respiratory infection. The diagnosis can be made by a number of enzyme immunoassays (Rotazyme) and latex agglutination tests with good specificity and sensitivity. Stools collected early in the course of the illness are more likely to contain virus, than those collected 8 or more days after the onset of illness. Treatment is directed toward correction of dehydration with oral fluid replacement. Breastfeeding is the most important and available preventive strategy, because human colostrum contains rotavirus antibodies.

Adenovirus commonly causes a wide range of human diseases, including conjunctivitis, pneumonia, and upper respiratory infections. A subgroup of adenovirus (enteric adenovirus) has been found to cause acute gastroenteritis, lasting 4 to 20 days. After rotavirus, enteric adenovirus is the next most common cause of viral gastroenteritis in infants and children, accounting for 5% to 10% of hospitalizations for acute gastroenteritis in children. They have been linked to outbreaks in child care centers, and asymptomatic excretion can occur. Most episodes occur in children less than 2 years. Infection increases in the summer months. Symptoms are indistinguishable from those associated with rotavirus but are less severe. The diagnosis is a presumptive one. Research laboratory confirmation can be made by solid phase immunoassays, electron microscopy, and viral culture but these studies are not routinely
Gastroenteritis and Dehydration

Dehydration

Defining and Classifying Dehydration

Dehydration results from acute vomiting and diarrhea. Diarrhea is the most common cause of dehydration in infants and children, and is a leading cause of death worldwide in children less than 4 years of age. Quantifying the degree of fluid loss by history and the amount and type of fluid intake can help to determine dehydration severity and the risk of electrolyte imbalance. The amount of urine output and the presence or absence of tears as well as the presence of documented weight loss, can help determine the severity of dehydration present. Other important aspects of the history include the presence of fever, sweating and hyperventilation, which may cause insensible losses, contributing to the degree of dehydration.

Clinical criteria can be used to estimate degrees of dehydration. The presence of fever, irritability, tachycardia, tachypnea, and the absence of tears, sunken or not, are all indicators of dehydration. The absence of tears, sunken or not, is an important indicator of dehydration. It should be performed in a warm room. Light pressure is applied to the finger nailbed. And the time from blanching to restoration of color to the nailbed is measured. It may be preferable to assess capillary refill centrally (over the chest or forehead) and peripherally (fingers), so that the two can be compared. A delay of less than 2 seconds is normal. Delays of 2 to 3 seconds may indicate moderate dehydration, and more than 3 seconds in delay may indicate severe dehydration. Most children with clinically significant dehydration, will have 2 of the following 4 clinical findings: 1) capillary refill greater than 2 seconds, 2) tacky mucous membranes, 3) no tears, and 4) ill appearance.

Management

The decision to hospitalize or to attempt outpatient management will be based on the clinical findings, combined with a history of fluid intake, the frequency of urination, assessment of concurrent stool losses and the response to therapy.

Once a child is presumed to be dehydrated, the degree of dehydration needs to be determined. Acute weight loss can be used to determine the degree of dehydration, but accurate baseline weights in growing children are almost never known. Clinical criteria can be used to estimate degrees of dehydration. Mild dehydration is 5% or less, moderate is about 10%, and severe dehydration is about 15% or greater. This classification is relative and not well standardized. If severe dehydration or uncompensated shock is present, the patient should be immediately treated with an IV fluid infusion (20 cc/kg of normal saline or lactated Ringer's) to restore the intravascular volume. It is likely that more than one fluid bolus may be necessary to restore the patient's intravascular volume, since 20 cc/kg only corrects 2% of the body weight. Therefore, the patient should be reassessed after each fluid bolus.

Children with mild to moderate dehydration can be initially treated with oral rehydration. Contraindications to oral rehydration therapy (ORT) include severe dehydration, intractable vomiting, and severe gastric distention. Children who had initially received IV fluid infusions for severe dehydration who now feel well enough to take oral fluids should also be considered for oral rehydration. Oral rehydration therapy (ORT) can be as effective as IV therapy. It is noninvasive and inexpensive. The AAP recommends that rehydration solutions contain 70-90 mEq/L of sodium, 20 mEq/L of potassium, and 2.0-2.5 grams/dL glucose. Examples of rehydration solutions (ORS) are: Rehydralyte (70mEq/L Na, 20 mEq/L K, 2.5% glucose, and the WHO solution (90m Eq/L Na, 20 mEq/L K, 2% glucose). ORT should initially be given in small, frequent volumes, 5 to 20 cc every 5-10 minutes, and advanced slowly to approach 5 cc/min. The degree of dehydration and the presence of ongoing losses dictate the volume of fluids to be

Water and Electrolyte Balance

Electrolytes and acid-base balance play a key role in the regulation of fluid balance. The main electrolytes involved in fluid transport are sodium, chloride, potassium, bicarbonate, and calcium. The balance of these electrolytes is critical to maintain fluid homeostasis. An alteration in the balance of any of these electrolytes can lead to dehydration or dehydration-related complications.

Acid-base balance is also important in fluid balance. The acid-base balance is regulated by the respiratory system and the kidneys. A disturbance in acid-base balance can lead to dehydration or dehydration-related complications. For example, respiratory alkalosis can cause dehydration, while respiratory acidosis can lead to dehydration-related complications.

Gastroenteritis

Gastroenteritis is a common cause of dehydration in infants and children. The most common cause of gastroenteritis is rotavirus, which is associated with 30-40% of gastroenteritis cases in children. Other common causes of gastroenteritis include norovirus, adenovirus, and cytomegalovirus.

The incubation period for rotavirus is 12-48 hours, and the illness typically lasts for 3-5 days. Symptoms include vomiting, diarrhea, fever, and dehydration. Treatment is usually supportive, including replacement of fluids and electrolytes.

Diagnosis of gastroenteritis is often based on clinical presentation, but laboratory tests such as stool analysis and serum electrolytes may be used to confirm the diagnosis.

Treatment of gastroenteritis is usually supportive, including replacement of fluids and electrolytes. In severe cases, hospitalization may be necessary for fluid and electrolyte replacement.

Prevention of gastroenteritis is important in reducing the incidence of dehydration in infants and children. Prevention strategies include good hand hygiene, proper food handling and preparation, and proper disposal of waste.

Caliciviridae

The Caliciviridae family is another group of viruses that cause gastroenteritis. The family includes several genera, including Norwalk-like caliciviruses, Sapporo-like caliciviruses, and swine-like caliciviruses.

Norwalk-like caliciviruses (NLVs) are responsible for a significant proportion of gastroenteritis outbreaks. The incubation period is typically 1-4 days, and the illness lasts for 2-3 days. Symptoms include vomiting, diarrhea, fever, and dehydration. The diagnosis of NLVs is usually based on clinical presentation, but serology may be used to confirm the diagnosis.

Sapporo-like caliciviruses (SLVs) are another group of caliciviruses that cause gastroenteritis. SLVs are associated with a milder form of gastroenteritis than NLVs. The incubation period is typically 2-4 days, and the illness lasts for 1-2 days. Symptoms include vomiting, diarrhea, fever, and dehydration. The diagnosis of SLVs is usually based on clinical presentation,

In conclusion, gastroenteritis and dehydration are common problems in infants and children. The diagnosis and treatment of gastroenteritis and dehydration require a thorough understanding of the underlying mechanisms and appropriate management strategies.
administered. If the degree of dehydration is mild (3-5%), the volume of ORS administered should be 50 cc/kg (i.e., 5% of the body weight) over 4 hrs. Those with significant dehydration (5-10%) should received 100 cc/kg (i.e., 10% of the body weight) of ORS over 4 hours. In either case, an additional 10 cc/kg should be given for each diarrheal stool seen. Once rehydration is complete, maintenance fluid is given. Examples of maintenance oral solutions are: Pedialyte and Infalyte, containing 45-50 mEq/L Na, 2-2.5 mEq/L K, 1.5-2.5% glucose.

Patients with mild dehydration can potentially be managed without laboratory analysis. However, in moderate or severe dehydration, laboratory studies should be obtained to look for electrolyte abnormalities of to measure the degree of metabolic acidosis.

Children who are severely dehydrated and those who cannot retain oral fluids because of intractable vomiting should be hospitalized and treated with IV fluid. Once the initial resuscitation phase is completed, replacement IV therapy should be instituted, taking into account fluid and electrolyte deficits as well as ongoing losses. Usually, half of the replacement therapy in addition to the maintenance fluid requirement is given over the first 8 hours, and the second half is given over the next 16 hours. However, patients with hypernatremic dehydration (serum sodium >150mEq/L) require special intervention. After initial management with normal saline or lactated Ringer's, the replacement fluid is given more slowly, over 48 hours or more. This is done because rapid correction of hypernatremia can result in acute brain swelling, brain herniation, and death. Therefore, care should be taken to avoid dropping the serum sodium by more than 15mEq/L per 24 hours.

Once a child is adequately rehydrated, the question of when to start feedings arises. It was previously perceived that a period of "gut rest" should follow rehydration of patients with acute gastroenteritis. However, numerous trials have shown no advantage to this strategy. The concept of early refeeding is replacing the old concept of "gut rest". Numerous trials have shown that early feeding of age-appropriate foods results in faster recovery. Breast fed infants should continue nursing despite diarrhea. Following rehydration, children with mild diarrhea who drink milk or formula can tolerate full strength feedings. The traditional BRAT diet (bananas, rice, applesauce, toast), although acceptable, should be considered to be a concept representative of a bland diet rather than a specific diet. Controlled clinical trials have shown that starches, complex carbohydrates (rice, wheat, bread, potatoes, cereals), soups, fresh fruits and vegetables, yogurt, and lean meats are better choices, and well tolerated. Fatty foods, juices, teas, sweetened cereals, soft drinks, are poor choices, and should be avoided. Some patients may benefit from lactose-free or low-lactose formulas.

Most pediatricians and experts recommend against using anti-diarrheal agents such as Imodium (loperamide), Pepto-Bismol (bismuth subsalicylate), and Kaopectate. This is more of a precaution since many studies do show some beneficial effects from these medications in patients with mild diarrhea. However, patients with mild diarrhea will get better on their own so these medications are usually not necessary. For young children with severe gastroenteritis, there is insufficient data to confirm the benefit and safety of these medications, which is why they cannot be recommended routinely at this time.

Questions

1. 1. Which diarrhea causing organism may be also cause neurologic symptoms?
2. 2. What is the most common viral cause of acute gastroenteritis, and what are its associated symptoms?
3. 3. How is Giardia lamblia most easily diagnosed and how is it treated?
4. 4. List 4 physical signs of dehydration in children?
5. 5. How are children with mild dehydration initially treated?
6. 6. How are children with severe dehydration initially treated?

Answers to questions

1. Shigella.
2. Rotavirus. It causes fever, vomiting, and watery diarrhea.
3. The diagnosis can be made by antigen detection, identifying cysts in the stool, endoscopy or examination of jejunal contents. It is treated with metronidazole or furazolidone.
4. Sunken fontanelle, absence of tears, sunken eyes, sticky/tacky oral mucosa, delayed capillary refill, reduced skin turgor, inactivity/lethargy, tachycardia, hypotension.
5. With oral rehydration, small frequent volumes 5-20cc every 5-10 minutes, advanced slowly.
6. With IV fluid infusion of normal saline or lactated Ringer's at 20cc/kg. Oral rehydration with ORS is commonly employed in other countries.
Case #1: At her one month well child visit, worried parents ask about their child's protuberant abdomen. She had been breast-feeding well during the first week, but her intake has been declining and she has begun spitting up. Physical examination finds lethargy, pallor with diaphoresis, tachycardia, distended loops of bowel, and rectal examination finds a narrow anus, and further insertion gives the impression of putting on a glove two sizes too small. The narrow canal extends for two centimeters, then widens into a pool of loose stool. When the examining digit is withdrawn, it is followed by a sudden spurt of particularly foul-smelling stool laden with mucus and streaked with blood, accompanied by a moderate amount of flatus. Questioning the parents identifies the failure to pass stool or flatus without stimulation with a rectal thermometer, having received instruction to do so from her aunt who is a nurse.

An abdominal series is obtained which demonstrates dilated bowel loops and a pattern resembling an acute bowel obstruction. Hirschsprung's disease with acute enterocolitis is suspected.

Constipation is a commonly used term, but its definition is somewhat ambiguous. It could refer to conditions such as: a) the stools are hard, b) the stool is difficult or painful to pass, 3) no stools for a period of time, 4) a bloated feeling, 5) painful cramps associated with a segment of stool that is not moving well, 6) a chronic condition in which a patient's stooling frequency is less than average. All of these definitions are used in medical and/or everyday communication, but it is preferable to use specific terms to describe the symptoms of the patient. The specific findings and their clinical significance will be described in this chapter.

Enterocolitis (as seen in case #1) is the extreme sequel of fecal retention, and is almost unique to Hirschsprung's disease, itself a uniquely pediatric version of the broader definition of chronic constipation: "a delay or difficulty in defecation, present for two or more weeks, sufficient to cause significant distress to the patient" adopted by the guidelines of the North American Society for Pediatric Gastroenterology and Nutrition (NASPGN). The subject is best broken into two broad categories: infants and children.

Infantile constipation: Per the guidelines, this does not include neonatal delays in defecation since the structural anomalies (imperforate anus, cloacal exstrophy, and other perineal anomalies, as well as intestinal atresia, stricture or web, volvulus, duplication, or perforation) and genetic diseases (e.g., meconium ileus of cystic fibrosis) often present in the first few days. Newborns should pass their first meconium stool within 24 hours. Those who don't have a higher risk of GI conditions associated with constipation. However, this criterion should not be relied on in isolation since pathologic conditions will not necessarily present this way. The algorithm proposed by the NASPGN constipation subcommittee emphasizes early suspicion of serious disease, by rapidly sorting out newborns with delayed passage of meconium for rectal biopsy and directing infants with "fever, vomiting, bloody diarrhea, failure to thrive, anal stenosis, tight empty rectum, impaction and distention" to immediate further evaluation, including subspecialty consultation as needed.

The workup begins with a thorough history and physical examination. The above alarm indicators are searched for, as are signs of other structural anomalies. The rectal examination is key, with careful assessment of the anal location, anal neurologic function (the anal wink, which assesses both the sensory afferent and motor efferent pathways), anal structure (looking for distention of the internal anal sphincter), anal tone (looking for spasticity or patulousness), function of the muscles of the pelvic floor (which provide additional help with control of defecation), and rectal diameter and tone (looking for signs of chronic distention even if no stool is present on the day of exam). The anal location should be halfway between the posterior border of the scrotum or posterior fourchette and the tip of the coccyx. Anything outside of the middle third of this region should raise the suspicion for a "perforate imperforate anus" (a structure resembling an anus is visible externally, but it is not contiguous with the rectum). If benign constipation is found, treatment is stratified based on age and developmental state.

Exclusively breast fed infants are permitted a longer interval between stools if they show no signs of distress or distention and if they are not prone to becoming impacted.

In exclusively formula-fed infants, my favorite strategy is the substitution of a commercially available partially hydrolyzed formula, which may produce suitable loosening of the stools. Malt soup extract (a dehydrated powder derived from an effusion of malted barley used in the brewing industry) has been advocated by the committee, as have corn syrup, lactulose or sorbitol, while the use of mineral oil was cautioned against due to the risk of aspiration posed by the frequency of gastroesophageal reflux and
Impaction is most commonly dislodged by glycerin (non-stimulant) suppositories for which the commercially pre-softened versions sold in soft plastic applicators (glycerin gel) have been my personal favorite, as they provide more immediate relief (the traditional refrigerated suppositories require a wait while they melt in situ). Stimulant enemas are to be avoided in young infants.

Older infants who are of an age where pureed foods would be appropriate should have the fiber content of their diet optimized (i.e., push fruits and vegetables and reduce the other starches). Another personal favorite in the older formula fed infant is the use of undiluted apple juice (not apple drink) for its sorbitol content, titrating the amount administered to the stool texture while making certain that formula intake remains adequate. Pear and prune juice can also be used as they are high in sorbitol, but the cost of the former and the TASTE of the latter are often limiting factors.

Case #2: This 6 year old male presents with fecal soiling on a daily basis, which began in late October. He claims he "can't tell when" he is about to soil. His parents report multiple bouts daily of fecal urgency where he rushes to the toilet, only to pass small amounts of diarrheal stool. His toilet sitting behavior is peculiar in that he sits far back on the toilet seat with his knees extended and his toes pointed, straining at defecation. Once or twice weekly he will pass a very large caliber formed stool, which has on occasion plugged the plumbing. This pattern was not thought to be a problem by his parents as it began shortly after they began potty training him at two years old so that he could enter preschool earlier than rest of the neighborhood children. The dietary history finds that he eats the school breakfast and lunch, and will often not touch his vegetables at supper. Closer questioning indicates he does not pick fruit or vegetables from the salad bar at school, and the school typically offers only sweet buns or a burrito for breakfast. Physical examination finds a midline mass in the lower abdomen, with a rectal examination that shows a normally placed anus with an intact anal wink and a perineum coated with stool. The anus is shortened with the internal anal sphincter dilated by a massive boule (little football) of formed stool. You are unable to accurately assess the diameter of the rectum as the stool appears to fill the pelvic bowl. The stool tests negative for occult blood.

Unlike the child with Hirschsprung's disease in the first illustration, the retention of stool in the older child who does not have a structural or neurogenic anomaly (as seen in case #2) will NOT cause secondary inflammation and enterocolitis, regardless of the duration of the problem. This lack of inflammation is an important differentiating factor that permits immediate identification of the older child with chronic constipation. The primary cause is voluntary fecal withholding, usually due to fear of pain on defecation, giving rise to the term "Psychogenic Constipation". The often accompanying overflow diarrhea or involuntary soiling arising from passage of looser chyme above and around the impaction is termed Encopresis in verbal analogy to enuresis. In simpler terms, the child has a football shaped mass of hard stool in the rectum which reduces the sphincter's ability to hold in liquified stool (chyme) coming from above, which results in soiling. The withholding behavior most often arises from a pattern of passage of large caliber stool as was the case with our illustration, but it can arise in response to a single traumatic event, such as a particularly large stool resulting in a traumatic fissure, a too-rapid transition from diarrhea with a raw perineum to fully formed stools, perianal cellulitis (more properly erysipelas, an intensely painful superficial infection of the anus and surrounding structures with Group A streptococcus identifiable by culture of the affected area), or least frequently but most insidious: overt trauma of physical or sexual abuse.

As in infantile constipation, the history and physical exam are key. The above historical markers are useful in establishing an understanding of the process by the patient and his or her caregivers. Dietary issues must also be explored, as well as the pattern of toileting (it is amazing how little time and opportunity school age children seem to have for sitting on the toilet, with some schools having policies of allowing only two minutes per bathroom break).

The issues on the physical examination of the older child are the same as those of the infant, particularly those regarding the rectal examination. Indicators of failure to thrive are more important beyond the first year, since celiac disease and cystic fibrosis occasionally present with constipation instead of diarrhea, and Crohn's disease can leave the rectum fully capable of extracting fluid from the reduced flow of chyme arising from the reduced appetite, if the inflammation is confined to the small bowel or proximal colon. Hypothyroidism is a particularly rare (but often cited) cause of constipation. A particular caution regarding Hirschsprung's disease bears noting as a significant fraction of the cases present beyond the second year of life in children who require stimulation to trigger defecation: repeated suppositories and enemas will often dilate the spastic segment making it impossible by digital examination alone to identify what should otherwise have been a microcolon. If suspicion is high (inability to spontaneously pass flatus or a strict requirement of stimulation to pass stool which when triggered tends to be foul, loose, and voluminous), an unprepped barium radiographic colon examination is indicated. This study should specifically look for a transition zone, to and fro peristalsis in the unobstructed segments, or uniform mixing of the contrast material throughout the colon (rather than concentration of the remaining barium in the rectum) on the 24 hour delayed film (hence the stipulation for barium rather than water soluble contrast which would tend to be absorbed by the next morning). If the radiographic study is equivocal, anorectal manometry may be of benefit. If either are indicative of
Constipation

Hirschsprung's disease, the diagnosis is confirmed by biopsy of the rectum deep enough to include the myenteric plexuses, as their absence indicates the disease.

If simple constipation without impaction or soiling is identified, therapy begins with education regarding the need for a more regular defecation pattern to prevent progression of the problem.

Dietary intervention is advocated, emphasizing fiber and fluid in accordance with proper nutritional guidelines. Here I find a concrete set of recommendations is most helpful in facilitating compliance, and I have abridged the USDA's food pyramid to a set goal of 6 servings of fruit or vegetables daily with a like number of servings of fluid, which is even further simplifiable to 2 servings of fruit or veggies at each meal which is easily understood by preschool AND adolescent patients.

More importantly, the need for regular toileting in the already potty-trained is emphasized, and I ask that they sit on the commode twice daily after meals to take advantage of the gastrocolic reflex to promote more regular rectal emptying. As in our illustration above, there must be an immediately preceding meal for the process to be most effective, and I have found that eating two fruits before toileting to be helpful. Suppers eaten out should be followed by a trip to the restaurant toilet to avoid missing the increased post-prandial peristaltic activity. A five minute time limit is set for commode sitting to avoid any sense of a punitive nature to the requirement and in some cases I will advocate using a kitchen timer in a "beat the clock" game if appropriate for the patient's personality.

Encopresis on the other hand is an indicator of repeated impaction, and usually is accompanied by enough dilatation as to render the rectal musculature patulous. Here again, education is key, and to simplify the biophysics (the wall tension is proportional to the fourth power function of the bowel lumen diameter), a quick analogy to a balloon that has been repeatedly inflated to the point of flaccidity is readily within the experience of most 4 or 5 year olds. Likewise an analogy to repeatedly compacting the trash over a 3-4 day period rather than dumping it daily will usually trap a kindergartner into admitting such behavior is likely to lead to a heavier, harder and bigger trash bag (and stool). Most importantly, education and discussion is important which should center on the cycle of pain at defecation leading to withholding which results in larger, firmer stools which in turn leads to more pain at defecation, perpetuating the cycle. This helps create understanding in the patient and the parent as to the origin of the process and its ultimate eradication. A thorough discussion of the mechanics of impaction and overflow passage of the as-yet unformed stool around the obstruction helps explain why distention of the rectum and internal anal sphincter and distortion of the levator structures of the pelvic floor result in inadvertent passage of loose stool whenever voluntary control of the external anal sphincter is relaxed. A thorough understanding is important in defusing the animosity that often arises between the patient and caregivers (parents, school, babysitters, etc.) over misunderstanding of what causes and perpetuates the soiling.

Treatment in the impacted, encopretic patient starts with disimpaction. High dose mineral oil and polyethylene glycol bowel preparation solutions have demonstrated efficacy and magnesium citrate, lactulose, sorbitol, senna and bisacodyl having been used anecdotally. Though the NASPGN subcommittee found that the oral route can be effective, typically this route is messy and more time-consuming. I strongly prefer a series of hypertonic phosphate soda enemas that are administered at 12 hour intervals. Typically only 3 are required, but the importance of removal of all formed elements is emphasized to prevent worsening the overflow diarrhea in the face of the fecal softening to follow. Caution is advised in using too much or too many enemas as each leaches a substantial bolus of calcium. In the case of particularly large and firm impaction, pre-softening by application of a mineral oil enema an interval before the stimulant one can be helpful. Saline enemas were also advocated by the committee as safe and effective, but soap suds, tap water and magnesium enemas are discouraged due to toxicity.

The next step is fecal softening, the issues being two-fold: produce a stool loose enough to be eliminated by the patulous rectum, AND eliminating any association of pain with defecation. Again, while the committee found lactulose, sorbitol, magnesium hydroxide, magnesium citrate, and mineral oil to be effective, I strongly prefer mineral or starting at 2-3 ml/kg/day but specifically titrating the dose to achieve the desired stool texture which I specify as "pancake batter", which has enough form to be routinely retained by the internal anal sphincter yet which is loose enough to empty out of the rectum with little more force than that of gravity alone whenever the levator structures of the pelvic floor are lowered and the anal sphincters are opened. In most cases, a patient whose rectum is dilated enough to allow soiling will have trouble expelling stool even the texture of toothpaste, which is the softest that can routinely be expected from fiber and fluid alone. A looser stool is needed to start the process, and mineral oil provides the cheapest and least flatulent method of attaining that goal. While the committee also made provisions for short-term addition of laxatives to this regimen, I feel anyone whose rectum is patulous enough to require such additional assistance, should have subspecialist evaluation, as this is by far the exception rather than the rule.

The third step is effective toileting: the already potty-trained patient should be seated on the commode with good foot support (to obviate any tendency to use the musculature of the buttocks and legs to assist in further withholding activity) on the commode twice daily after meals under the same
guidelines and for the same reasons as outlined in the simple constipation as above. The sitting is made "non-negotiable" simply to ensure its application as it will become the most enduring and important part of the regimen as the weaning process progresses. Those who are not yet potty-trained are excused from formal sitting but are encouraged to crouch in diapers after meals in an analogous fashion.

Once a better than daily bowel habit is established and withholding is clearly extinguished, weaning off the mineral oil can begin. It is taken VERY slowly, in part to avoid recurrence of pain and resumption of withholding, but more to allow time for the patulous rectum to regain motor tone. I illustrate the importance of this to the patient and family by referring back to the balloon illustration, pointing out the difference between inanimate latex and living muscle, which can regain tone and function. I specifically warn that the process will take months to improve, and that prolonged use of mineral oil has been proven benign. This helps improve adherence to the long-term nature of the measures involved, and weaning typically occurs at monthly intervals, and then ONLY if the rectum is indeed shrinking in diameter (and improving in function) and if the withholding remains extinguished. Failure with either issue should result in either maintenance at the current step or return to the next higher one.

Adherence to the mechanical measures involved typically results in an immediate return to continence with the completion of disimpaction, as the nondistended internal anal sphincter is able to retain the loose stool. Continued adherence to the slow weaning typically results in return to long term function (and confidence) through the months of steady increase in stool texture. Permanent adherence to a daily defecation pattern results in long-term avoidance of reimpaction, and is the ultimate goal of the process. Each step along the way involves the physician acting as coach, cajoling and encouraging patients and caregivers, solving problems in techniques, and refereeing any residual conflicts. It must be kept in mind that control in this issue lies with the patient. There is nothing we can (or should) do that will force regular toileting, and there are times when I have to call a "time out" from the process to enable the patient to proceed on his or her merry way until THEY are ready to work on the problem. I often remind parents that the only thing one will die of with routine encopresis is embarrassment, remembering that children are often beaten to death by caregivers for soiling behavior. As can be seen above, the initial visit to address the issue of encopresis can be particularly time-consuming, not with regard to the history or physical examination, but because of the need to impart the understanding of the process of the disease that will encourage an apprehensive child to undertake the measures needed to control in this issue lies with the patient. There is nothing we can (or should) do that will force regular toileting, and there are times when I have to call a "time out" from the process to enable the patient to proceed on his or her merry way until THEY are ready to work on the problem. I often remind parents that the only thing one will die of with routine encopresis is embarrassment, remembering that children are often beaten to death by caregivers for soiling behavior. As can be seen above, the initial visit to address the issue of encopresis can be particularly time-consuming, not with regard to the history or physical examination, but because of the need to impart the understanding of the process of the disease that will encourage an apprehensive child to undertake the measures needed to clear it. The hour rapidly fills with illustrations and instruction, and does not readily fit into a routine sick-child office visit. Time must be set aside for proper handling of the process, and I know most consultations for encopresis arise from the inability to carve out such time in the primary care practice setting.

Questions
1. The nurse points out a two day old healthy term infant who is otherwise ready for discharge who still has not passed meconium. Your next step is:
   a. Order a suppository prior to discharge.
   b. Careful physical examination, including digital rectal examination.
   c. Give a normal saline enema to prep for a barium enema.
   d. Call radiologist to discuss an unprepped barium enema.
   e. Rectal biopsy.
2. The exam of a 3 year old with recurrent impaction is normal except for the impaction and the absence of an anal wink. Which of the following are true.
   a. An anal wink is not commonly found in this age group.
   b. The anus may be so traumatized by the impaction that the wink cannot be reliably elicited.
   c. There may be a neurogenic component to the problem in addition to the psychogenic one.
3. Your examination of a chronically soiling 13 year old female finds a normal sized rectum containing soft stool. Is this routine encopresis?
4. A 6 month old infant has been getting suppositories and enemas every 3-4 days because she does not otherwise defecate. The stools were passed without apparent trouble on breast feeding. Rectal examination finds a normal sized rectum as far as you can reach. Does this rule out Hirschsprung's disease?
5. The barium enema performed yesterday was read as normal, but the remaining barium did not pass overnight. You obtain a followup film this morning, and find dilute barium evenly distributed from the cecum to the rectum. What is the likely diagnosis and why?

Answers to questions
1. Answer d is correct, and the radiologist will appreciate the warning as to why the exam is being requested without prior bowel cleanout (which may otherwise be performed as part of the radiology routine, rendering the same end result as answer c). Answer a will not only miss the diagnosis but may also render diagnosis more difficult later if the pattern is set for stimulation for defecation. Answer b may give the diagnosis if a microcolon can be identified on exam, but can
make interpretation of a barium enema difficult. Answer c is wrong for the same reasons as a and b. Answer e is doing too much too soon.

2. Correct answers are both b and c. Anal winks can be expected at any age unless the anus has indeed been badly traumatized. Its absence usually indicates a neurogenic component, and the examiner is prompted to carefully assess the tone of the sphincter and retrospectively look for other signs of aberrant function of the longer neuron sensory and motor tracts or signs of sacral anomalies. If the issue is still in doubt, it can be deferred by one visit. The process can still be addressed by full fecal softening and re-establishment of regular bowel habits since the therapies diverge at a later stage where a timing suppository needs to be added to maintain regular defecation as the weaning progresses and the stool becomes firmer. Full fecal softening is needed initially for both causes to address the flaccidity of the rectum.

3. No, the absence of impaction is worrisome, and the behavioral and social history are likely incomplete. The above pattern suggests voluntary soiling, in which a socially uncomfortable behavior is expressed to avoid an even more uncomfortable behavior, such as sexual abuse.

4. NO! The enemas may have dilated the rectum beyond the reach of the examining digit, and it is common for patients with short segment Hirschsprung's disease to pass the softer stools of breast feeding but have trouble with formula and pureed food. Expert radiographic evaluation is necessary, and the assistance of a pediatric surgeon or gastroenterologist may be helpful.

5. This is the typical appearance of the delayed view in a patient with Hirschsprung's disease. The obstruction is of high enough a grade that the portion of the colon with normal ganglion innervation has set up a "to and fro" pattern of peristalsis, evenly mixing the remaining barium with the increased fluids present in the lumen, rather than transporting the barium to the rectum where the excess fluid is removed (which is the appearance of the normal colon).

Gastrointestinal Bleeding and Peptic Ulcer Disease

Upper GI Bleeding

Case 1: The parents of a 3 year old who you have been following since birth for biliary atresia, call to report a "nosebleed" (epistaxis) overnight. Closer questioning discloses that what they are calling a nosebleed is simply a puddle of blood found on the pillow. Having anticipated this potential complication, you ask them to meet you in the Emergency Department. There you find him to be in no distress, with no tachycardia or diaphoresis. You can find no site of bleeding in the nose or pharynx, and you also note his ascites has disappeared and his spleen seems smaller than when you saw him last week. What's going on?

Case #1 described above illustrates the one exception to the rule in large volume bleeding. In children with varices and ascites (both arising from portal hypertension) the acute volume loss from the bleeding can be repleted by 'auto-infusion' of the ascites. Portal hypertension triggers ascites at relatively low pressures (10-12 mm Hg), and the volume depletion from bleeding results in enough reduction in the portal pressure to coax the fluid back into the circulation. The hypovolemic state accounts for the loss of the previously existing splenomegaly. These patients also illustrate that all blood loss is whole blood and that the hemoglobin and hematocrit will not fall until they are volume repleted with crystalloid or plasma. Cirrhotic patients with ascites are the only ones where the acute CBC may be a better indicator of the volume lost than vital signs at presentation.
His hemoglobin is 70 (hct 21). His INR is 1.2. He continues to be in no acute distress and he is able to go directly to endoscopy, having requested 2 units of packed RBCs to be available in the OR for emergency transfusion if needed, with more packed RBC and fresh frozen plasma standing by. A small ulcer is found in the distal esophagus, with an associated gastric varix. There are two other fully distended esophageal varices which are band ligated, and while sclerotherapy is considered for the gastric varix (the banding is impossible to accurately apply in this location) you elect to watch as it appears to be thrombosed and plans are made to return for a repeat endoscopic inspection and treatment as needed in a week or two. He tolerates the procedure well with no complications, and after talking with his parents, you call his transplant specialists to update them on his situation.

Gastrointestinal bleeding covers a wide topic, and is best managed by subdividing it into smaller and smaller entities. But even before making the first obvious decision as to whether it’s “upper” or “lower” GI bleeding, the first step is to make a rough assessment as to how MUCH bleeding is going on. In pediatrics, the best single vital sign for assessing acute volume depletion is the heart rate rather than the blood pressure, since infants, children and adolescents have a huge reserve capacity for increasing cardiac output by increasing the heart rate. Thus, the blood pressure begins to fall only in late shock. Orthostatic change in the heart rate is a useful sign (only occasionally unreliable), since a difference of 10% or more may indicate substantial acute volume depletion. Another sign to look for are cool extremities, often with a relatively sharp demarcation between cool and normal skin temperature, as an indication of peripheral vasoconstriction. These signs are applicable to acute volume depletion from any cause (such as vomiting and diarrhea) and not just to acute bleeding. As with any pediatric life support issue, the first steps in assessment and management are to verify airway, breathing, and circulation (ABC). Acute volume depletion requires rapid volume replacement and determination of the source of loss.

When there is a significant difference between the degree of volume loss from either end and the apparent normal state of the intravascular volume, the next step is to verify the material as blood and that it is indeed coming from the patient. Here are some anecdotal examples: 1) A child whose bright red diarrheal stools are closer in color to the red-orange of the food dye of the fruit punch that he has been guzzling during his acute gastroenteritis than to any blood color you’ve ever seen (i.e., the “bloody” diarrhea is really fruit punch). 2) The robust, demanding, overfed infant whose mother has mastitis and whose hematemesis is actually coming from his mother’s blood loss (i.e., swallowing mother’s blood from a bleeding nipple) rather than his own. 3) The newborn whose mother had placenta previa and a particularly bloody delivery whose hematemesis again is ingested maternal blood and not his own. The first example often is resolvable during the initial phone conversation. The third can be identified reliably by performance of an Apt test for fetal vs adult hemoglobin, but the second often requires careful history taking and examination to exclude intrinsic GI bleeding.

Converse consideration must also be given to those who present with signs of acute intravascular volume depletion (especially impending shock) and NO signs of bleeding discernible externally. The GI tract can easily hide a loss of blood amounting to a substantial portion of the total intravascular volume. Processes with high rates of bleeding, particularly if beyond the stomach (making them less prone to hematemesis) must be considered. Such lesions include (but are not limited to) duodenal ulceration with arterial bleeding, duodenal varices or varices at small bowel anastomotic sites (in the case of children with surgical hepatoportoenterostomies), small bowel vasculitides, hemobilia (bleeding in the biliary tract), and enteric duplications (including Meckel’s diverticula) with secretory mucosa and secretory products that lead to ulceration.

The next step is to verify that the bleeding is indeed coming from the GI tract, in part to determine whether the following algorithms will be applied, but also as reassurance that the volume of any recurrent bleeding is not likely to be high or is likely to be easily managed. Epistaxis is a common cause for moderate volume bleeding (some epistaxis blood is swallowed and then vomited as hematemesis) and is more common than peptic ulceration as a cause of hematemesis and melena. Always check the anterior portion of the nasal septum for evidence of blood and ulceration indicative of bleeding. Application of direct pressure for 5 minutes (by the clock) to allow for good clot adherence and retraction (no peeking or the clot will lift off and the bleeding will resume) during any recurrent bleeding episode, is usually all that is necessary. Cautery typically causes more problems than it solves. Topical antibiotics can be used to treat nasal impetigo if that is the cause of the epistaxis. Dental and oral bleeding typically is smaller in volume and is usually identifiable on close inspection. Anal fissuring is technically a "GI" lesion, but the risk of rebleeding is low and its generally benign nature differentiates it from the lesions below. Here, the bleeding is typically bright red, and can be painless (though more often associated with pruritus at the anus or cramping prior to the passage of a larger caliber formed stool). Passage of clots is possible if the fissure extends internal to the internal anal sphincter. But the differentiating hallmark is any continued dripping of blood into the toilet after the formed stool has passed (and the internal anal sphincter has closed) or the persistence of bright red blood on the toilet paper for more than 2-3 wipes. Either of these indicates the presence of the lesion outside the internal (but potentially proximal to the external) anal sphincter. Good inspection requires gentle separation of the buttocks, best attained in the decubitus
position with the knees up against the chest. The goal is to open the external anal sphincter, often attainable by having the patient take a slow deep inhalation. The fissure is identifiable as the red or white-based linear ulcer, usually anteriorly or posteriorly positioned. Specific attention should be paid to whether the margins appear undermined (traumatic fissures have simple vertical margins, while the anal lesions of Crohn's disease and the vasculitides are caused by the undermining of the subcutaneous supportive structures).

Once it is verified the patient is indeed losing blood from their GI tract, but is stable, attention turns to specific diagnosis. Here again subdividing the possibilities is helpful, with the next step being the semi-artificial conceptual one of "upper" vs "lower" tract bleeding. With the provisos of the preceding paragraphs accounted for, hematemesis is a reasonable indicator of an UGI source. Conversion to "coffee grounds" is not necessarily indicative of a gastric source since any blood placed in acid for even a few seconds converts to acid hematin (brown color), and conversely if the acid is neutralized or is otherwise not present for any reason, the blood will not convert (and will be red). On the other hand, melena is only an indicator of bleeding in an area bathed in acid, and while this usually indicates the upper GI tract, other sites of acid production such as a Meckel's diverticulum (with acid secreting ectopic gastric mucosa) or occasionally acid fermentation in the right colon can trigger the same chemical conversion. And conversely, the absence of melena in the bleeding does not preclude an UGI site, since brisk bleeding from an arterial source in the duodenum, plus the rapid transit times of infants and younger children, and their lower acid secretion rates can result in passage of blood per anus that is still nearly bright red.

Quantification of the bleeding seen is of some help diagnostically, as is the presence or absence of associated symptoms and signs. Further stratification by age helps establish probabilities:

Newborns are not old enough (not enough time) to develop full peptic ulceration. Coagulopathy due to liver cirrhosis is possible, but rare. Neonates under the intense physiologic stresses that would place them in an NICU setting typically develop gastric erosions rather than deeper gastric ulceration or they can hemorrhage from DIC. Even vitamin K deficiency of infancy takes time to develop.

Infants have had time to develop some, but not all of the ills that befall older children. Again mechanical trauma to (or other lesions of) oral structures will result in small to medium amounts of hematemesis and are far more common than the erosions of severe reflux esophagitis or frank ulceration of the stomach or duodenum. The latter lesions are usually suspected by signs of dyspepsia, including (but not limited to) crying, irritability centering around feedings, colic, drooling, eructation (belching), and vomiting. The preverbal infant and young child often will not be able to adequately indicate the associated pain, and peptic disease is therefore more often not identified until there is significant vomiting or even frank hematemesis. Allergic gastroenteritis more commonly first appears in the first few months of infancy though eosinophilic gastritis can be identified at any age. Though more commonly presenting as failure to thrive, allergic gastroenteritis can occasionally present with hematemesis or other evidence of upper (and lower) GI tract bleeding.

In toddlers most of the above processes continue, but their added mobility increases the possibilities for mechanical issues, including ingested caustic agents (lye, tile cleaner, electric dishwasher detergent) or acids, and foreign bodies. A toddler presenting with signs of partial esophageal obstruction (inability or unwillingness to swallow solids) and intermittent hematemesis warrants a radiographic evaluation to establish the absence of a lodged foreign body and if no clear history is obtainable regarding of the duration of the lodgment, caution is to be exercised in its removal, since penetration of or even embedding in the wall of the esophagus or adjacent structures (e.g., vena cava or aorta) is not uncommon. Ingestion of button batteries is another special consideration in the case of foreign bodies since the lithium ones can retain sufficient charge as to cause significant mucosal burn even when they appear to be dead, while the mercury, silver, and alkaline button batteries usually do not.

In older children, a Mallory-Weiss tear (esophageal tear) or an erosion caused by prolapse of a portion of the gastric cardia (typically along the lesser curvature) is far more common than nonspecific gastritis, erosion (esophageal or gastric), or frank ulceration as a cause of UGI bleeding. Such bleeding usually follows a period of protracted and usually forceful vomiting, and is usually relatively limited in volume (a few teaspoons), but it can be profuse. A rectal examination with stool which is negative for occult blood helps verify the observed short duration of the process. Esophageal erosion typically is preceded by complaints identifiable as peptic in origin in pediatrics and is rarely present in a pain free setting, which requires an extensive burn. In the case of the older child or adolescent with this presentation, consideration needs to be given to NSAID-induced gastropathy arising from self-medication. The lesions are typically erosions arising from the disruption of mucosal cytoprotection due to the broader inhibition of prostaglandin synthetase. The incidence is low compared to the widespread use of these medications and the increase in the absolute rate of this complication reflects the increasing use of these medications in the over-the-counter setting.

**Upper GI bleeding etiologies by age:**

Age Small volume Medium volume Large volume
Newborn Gastric erosion Ingested maternal blood,
(in NICU setting) vitamin K deficiency, DIC.

Infant Mechanical trauma, allergic Same as infant, plus oral
or other gastritis, reflux lesions and ulcerations (rare).
esophagitis, ulcer.

Child Dental/oral source, Same as infant, plus Varices, arterial bleeding
hemoptysis, gastritis epistaxis, Mallory-Weiss from ulcer, DIC.
(non-allergic), tear/erosion, peptic ulcer.
reflux/chemical esophagitis,
(beware of foreign body).

Frank peptic ulceration remains uncommon as a source of bleeding during most of childhood and adolescence. Helicobacter pylori can play a role, and by itself, can cause bleeding from gastritis, though far more commonly it presents as a non-bleeding distinctive nodular gastropathy with preference for the antrum. While breath testing using labeled urea is identified as showing promise in establishing the diagnosis, the North American Society of Pediatric Gastroenterology and Nutrition's position is that endoscopy remains the only reliable means of establishing and refuting its presence, and that blood testing, due to poor specificity, is of little utility. In that regard, the low probability of Helicobacter gastritis in this age group even in the face of proven duodenal ulceration means that for each child appropriately identified by serologic testing, there will be several falsely labeled, and extension of this testing into evaluation of those who simply present with hematemesis or even just pain raises the likelihood of identifying false positives.

However, in true hemorrhage from the upper GI tract (defined as bleeding sufficient to require volume repletion with blood), peptic ulceration, variceal bleeding, and DIC are the most common causes. Of these, variceal bleeding is the most frightening, since blood loss rates can approach total blood volume within an hour or less. Varices arise from portal hypertension, which in turn arises most commonly from cirrhosis (e.g., biliary atresia as in case #1), but can also arise from extrahepatic obstruction of the portal vein. They most commonly present in the distal esophagus but may also be found in the gastric cardia or in the duodenum, where they can be far more difficult to treat. They represent an enlargement of the submucosalvenules as a means of rerouting the blood flow from the portal to systemic venous circulation (a porto-systemic shunt) and the degree of portal hypertension required to establish the shunt is only minimally higher than that which would produce splenomegaly or ascites, making these physical findings important in the evaluation of the hemorrhaging patient. Identifying patients at risk for this prior to hemorrhage is far preferable, and a routine search for these findings should be undertaken at every office visit of any patient being followed for a process that can lead to cirrhosis (such as biliary atresia in the case presented). The bleeding is typically painless, as the vessels are superficial to the muscular layers and the erosion that starts the bleeding is therefore particularly shallow. The initial (or "herald") bleed may be surprisingly small as the vein rapidly collapses and clots, but the dislodgement of that clot can be followed by particularly voluminous bleeding. Endoscopic examination for diagnosis and treatment either by banding or injection of sclerosing agents is the preferred acute management once volume repletion has made sedation or anesthesia possible, and while a trans-jugular intrahepatic portovenous shunt placement (TIPS procedure) may be palliative, the only "cure" is resolution of the underlying cirrhosis (by transplantation) for those with intrahepatic disease conditions. Those with cavernous transformation of the portal vein typically will create other intraabdominal shunts in locations which do not bleed so profusely if they can be carried into the second half of the first decade.

The myriad causes of upper GI bleeding prompt usage of a layered strategy to the diagnostic process. With the above history parameters and physical findings indicating that: 1) the patient is (at least temporarily) hemodynamically stable, 2) no clear site of bleeding external to the GI tract as an alternate source to explain the bleeding, 3) no known lodged esophageal foreign body, 4) medium to moderate bleeding in whom the presumed risk of later hemorrhage is worth giving serious consideration; the next step is placement of a naso-gastric catheter to assess the volume of blood that has not been regurgitated, and more importantly, to identify (or refute) any ongoing bleeding. A large-bore tube is recommended, as clots may need to be removed, and the orogastric (rather than nasogastric) route may facilitate evacuation. Gastric lavage with normal saline at body or room temperature is more comfortable. Icing of the saline is not required and in smaller patients, may produce significant hypothermia. Gastric sampling and lavage may be omitted if variceal bleeding is suspected and endoscopy is already planned, but the hemorrhaging patient typically otherwise deserves both procedures. The gastric sampling and lavage serves to identify the patient who has indeed bled briskly from the upper GI tract or who may still be bleeding, and who will need to go on to immediate endoscopic examination. In contrast, the patient whose bleeding is suspected as coming from a non-intrinsic or otherwise low-risk source does not require immediate endoscopy. A negative gastric aspirate in a patient
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who is otherwise only suspected of an upper GI bleeding site because of passage of melena and who has no hematemesis would prompt a search for alternate sites of acid-associated bleeding such as a Meckel's diverticulum.

Gastric aspiration will only rarely be falsely negative. This would typically occur in patients who are bleeding from a deep ulcer in the duodenal bulb with sufficient pylorospasm to prevent any regurgitation of blood into the stomach. These patients can easily be mistaken to have a Meckel's diverticulum with acid-secreting ectopic gastric mucosa and secondary ulceration as they too may present with volume depletion and melena but no hematemesis, and if suspicion is otherwise high (primarily due to tenderness to epigastric palpation) and/or larger volumes of bleeding, endoscopy is warranted anyway.

Endoscopy offers both diagnostic and therapeutic advantages, and typically is much more sensitive than radiographic evaluation of the upper GI tract as most hemorrhaging lesions are still superficial. Radiographic contrast studies are dependent on identification of a moderate-sized ulcer crater, and lack sensitivity in identifying risks for rebleeding such as a visible vessel or adherent clot. Even varices are difficult to identify radiographically, and endoscopy offers the ability to intervene to reduce the risk of rebleeding via variceal band ligation or intravascular injection of a sclerosing agent. Likewise, ulcers and other lesions at significant risk for resumption of hemorrhage may be addressed thermally or chemically through the endoscope.

Peptic ulcer disease is most accurately diagnosed by endoscopy. Referral to an endoscopist (usually a gastroenterologist) facilitates diagnosis and treatment since treatment regimens which consist of a cimetidine, ranitidine, etc.), proton pump inhibitors (e.g., omeprazole) and antibiotics for H. pylori create numerous therapeutic option combinations. Optimal therapeutic decision making for pediatric patients with peptic ulcer disease is best left to gastroenterologists who are most familiar with the most recent studies and recommendations.

If endoscopy fails to identify the bleeding lesion, further investigation of the hemorrhaging patient includes radionuclide scanning and angiography. Radiographic intervention can include selective intraarterial embolization as well as the TIPS (trans-jugular intrahepatic portovenous shunt) procedure, but a full discussion of this is beyond the scope of this chapter in basic diagnosis, as the choices are typically guided by the pediatric gastroenterologist involved in the endoscopic procedure above.

**Lower GI bleeding**

*Case 2: The parents of an otherwise robust 3 year old boy call with a frantic report of bright red bleeding per anus. Pain is denied, as are fever, malaise, or rash. The toilet bowl seems filled with blood and clots, but the anus wipes clean with one swipe and no further blood is seen. You are able to convince them to come to the office instead of heading for the ER. In the office, the child is in no distress and wonders what all the fuss is about. His vital signs are normal for age, and physical examination shows no abnormalities, including external inspection of the anus with the child in the knee-chest position on his left side to enable full exposure of the anus down to the internal anal sphincter. What do you do next?*

The patient who presents with bleeding only from the anus produces a separate (but overlapping) diagnostic tree. The presence of melena hints at an upper GI tract origin, but is simply indicative of passage of the blood through acid, which can arise in the presence of acid secreting ectopic gastric mucosa in any enteric duplication, of which the most common is a Meckel's diverticulum, or occasionally if the cecum is particularly acidic due to fermentation. Conversely the absence of melena does not exclude an upper GI tract origin if the transit time is sufficiently short or the acid is otherwise neutralized or not present and the bleeding is brisk enough.

As discussed in the patient who presents with hematemesis, the initial evaluation centers around rapid estimation of the volume of blood lost and the risk of ongoing or recurrent bleeding. Again the problem is best stratified into ages and rates of loss.

As with upper GI bleeding, the neonate has fewer diagnostic possibilities since most problems take a while to develop fully. While any infection (bacterial or viral) can lead to sufficient mucosal inflammation ranging from punctate bleeding to gross hemorrhage and DIC, necrotizing enterocolitis is almost unique to the neonatal period. In other respects, resembling ischemic injury in the older child or adult, the process in the neonate does more commonly include submucosalpneumatosis, implying compromise of the mucosal barrier. It usually presents with other signs of intestinal obstruction, partial or complete, and bleeding is typically one of the lesser findings, and is most commonly occult. It presents more commonly in the severely premature, but can afflict term infants who have a preceding clinical problem that predisposes them to bowel ischemia (such as polycythemia or birth asphyxia). Refer to the chapter on necrotizing enterocolitis.

Allergic enteropathy is more typically a problem of the young infant, as the inflammatory process is acquired and requires time to set up. It typically presents before 2 months of age with either occult or gross bleeding, and typically is accompanied by failure to thrive and/or a moderate degree of mucus in the stool to suggest widespread mucosal irritation. Involvement of the proximal GI tract (the stomach
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and small bowel) more commonly results in slow weight gain, signs of poor enteric motility (vomiting) and other signs of gastric stasis and protein losing enteropathy. Involvement of the colon is associated with mucousy stools laced with blood. In the latter, a Wright stain may be helpful only if it shows sheets of eosinophils, but a firm diagnosis rests on mucosal biopsy showing widespread nests of eosinophils in the submucosa rather than the scattered eosinophilia seen in more nonspecific inflammation. A clinical diagnosis may be made by rapid and complete resolution of the symptoms by elimination of the offending protein either by a return to exclusive breast feeding or substitution of a properly hydrolyzed formula (e.g., Nutramigen, Pregestimil, Alimentum). A switch to an alternate allergenic protein source (soy, goat’s milk, etc.) during a period of sensitization may result in further reactivity and only true hypoallergenic feedings are to be allowed. Widespread reports, laboratory research, and personal experience indicate that while generally hypoallergenic, maternal breast milk may contain identifiable fragments of cow’s milk protein from the maternal diet in quantities sufficient to trigger a reaction. Personal experience suggests the quantity needed in the maternal diet is substantial, and typically lies outside routine dietary parameters, however maternal exclusion of dairy products may be undertaken in the case of stubbornly persistent (and typically low-grade) inflammation. If allergic enteropathy (gastroenteropathy or colitis) is encountered, firm exclusion of the offending protein is to be undertaken for the entire first year of life in hopes of eliminating the clone of sensitized lymphocytes. This involves reading the ingredient panel of every item the child will eat, looking for “non-fat dairy solids” or “non-dairy” creamers (which contain powdered milk protein). If a soy allergy is present, the prohibition shifts to soy, including soy sauce and tofu. Typically the exclusion is not complete, and if (repeated) inadvertent exposure shows no sign of reaction, the restrictions can be lifted. But recurrent reactions can be severe if of the acute hypersensitivity (type I) variety. This can result in sufficient vomiting and diarrhea to cause significant volume depletion, and if uncertain as to the residual reactivity, a formal staged dietary challenge with nursing support (i.e., this may need to be done as an inpatient or in an observation unit) may be needed at one year of age before the offending food may be safely reintroduced in quantity.

Another cause for minor bleeding per anum that is unique to infancy is nodular lymphoid hyperplasia. It typically presents with punctate bleeding best characterized as streaks of blood with small streaks of mucus in otherwise normal stool in an otherwise thriving infant. The number of streaks and the amount of blood do not vary with fecal texture. This compares to infection, allergy or other more generalized inflammatory processes of the distal bowel where loose stool indicates inflammation, and therefore goes hand in hand with more mucus and blood. The only time the bleeding disappears in nodular lymphoid hyperplasia is in the face of liquid stools, in which case the streaks of mucus and blood are dissolved in the diarrhea but can be found by occult blood testing. Nodular lymphoid hyperplasia can readily be identified by proctoscopic examination which typically demonstrates a rectum that is studded with submucosal nodes measuring 2 mm across with central ulceration. The bleeding comes from the ulceration and the intervening mucosa is completely normal in appearance, explaining the disparity between the texture of the stool, the amount of bleeding and the normal growth of most of these infants. This permits exclusion of allergy and infection as possible causes since these typically cause more widespread inflammation, visible in the rectum of infants presenting with visible blood and mucus in the stool. Nodular lymphoid hyperplasia is a benign, self-limited process associated with the age-appropriate hypertrophy of the lymphatic tissue of the enteric submucosa. In some infants, the central portion of the overlying mucosa undergoes punctate ulceration. The cause of the process remains unknown. What is known is that the process normally becomes dormant during the latter half of the first year as the nodes regress in size (and activity), and though there may be occult blood found in the stool for the remainder of the first year, there is little likelihood of anemia and no association of any later enteric disease process. As such, my usual recommendation is to continue with routine feedings, introducing solid foods at the usual times as the process is not allergic in origin. The hemoglobin may be checked slightly more frequently than your usual schedule for age, and iron supplementation should be started only if it drops significantly. Inability to keep up with iron loss is atypical enough to warrant reassessment of the original diagnosis.

Toddlers in turn have a cause for chronic occult bleeding and often times severe anemia which is unique to their age range in overconsumption of milk. The lactoferrin of cow’s milk has an extremely high affinity for iron, higher than anything in the human iron transport system, and it typically is not saturated in milk as routinely sold (but it is saturated in infant formula). Lack of an alternate iron source and excessive intake of milk can result in severe iron depletion, and as iron is also required for maintenance of gut mucosal integrity, the process accelerates as the iron stores fall. Hemoglobins of 4.0 gm/dl are not uncommon in this setting, and if tested, the stools may be positive for occult blood. The diagnosis is made by a detailed diet history, and verification of the extremely low iron stores. The cure is effected by a return to a truly regular diet for age with reasonable milk intake and a sufficient source of iron, though an occasional patient will require transfusion.

Processes that can cause small but visible quantities of bleeding at any age are dominated by infection, though anal fissures are even more common, as described previously. The most common worrisome organisms include Campylobacter, Salmonella, Shigella, and enterotoxigenic E coli, but other routine pathogenic bacteria and protozoans can be acquired from contaminated food and standing water
sources. The first 4 are routinely included in culture screens for enteric pathogens (the rest are not). Campylobacter can cause a severe colitis which is identified more often now that CT scanning is the preferred method for identification of appendicitis in the patient presenting with crampy abdominal pain. The degree of thickening of the submucosa and muscular layers can be mistaken for transmural thickening indicative of Crohn’s disease, with the pain and bloody diarrhea adding to this diagnostic possibility. But a normal ESR in the proper clinical picture suggests a pause in the rush to colonoscopy and therapy until the culture results are available, since the process clears rapidly with erythromycin treatment. Shigella also warrants antibiotic therapy if found, and while treatment of Salmonella may raise the risk of producing a chronic carrier state, since most carriers arise from colonization of the gallbladder, cautious treatment with an agent concentrated in bile (such as trimethoprim/sulfamethoxazole) if the organism is sensitive may be warranted in the patient with ongoing or severe symptoms. The one organism whose treatment with antibiotics or antispasmodics is to be avoided is enterotoxigenic E. coli (such as E. coli O157). Use of these agents can produce enough enterotoxin release as to trigger Hemolytic Uremic Syndrome. Antibiotics should be held until the offending bacteria is positively identified, and even over the counter antispasmodic agents are to be avoided.

The immune suppressed patient presents a particular challenge, for in addition to the above agents one needs to consider atypical organisms such as cytomegalovirus and Mycobacterium aviumintracellulare (MAI), which can produce severe bleeding with transmural lesions scattered throughout the bowel and colon. Epstein-Barr virus can cause lymphoproliferative disease with chronic low-grade blood loss and more of a protein losing enteropathy picture. Typhilitis also occurs in the patient recovering from neutropenia as the new granulocytes are preferentially directed toward the inflamed cecum. These processes are rare in the immune competent patient.

Enterocolitis due to Hirschsprung’s disease can occur both prior to and after surgical repair. The latter instance is indicative of stricture at the anastomotic site and recurrence of functional obstruction. As a stasis phenomenon it can also be seen in those with ileorectal pouches and other anastomosis, and while it can cause bleeding, it usually presents with explosive, foul diarrhea. For evaluation of the infant presenting with enterocolitis as the first manifestation of Hirschsprung’s disease, see the chapter on constipation. Hirschsprung’s disease itself is covered in a separate chapter.

Other processes that can cause moderate bleeding volumes, usually as part of a broader clinical picture include general obstructive processes such as intussusception, volvulus, and other mechanical issues that can cause focal bowel ischemia. They usually present with other signs of obstruction, typically with an acute onset of crampy abdominal pain that cycles every 10 to 60 minutes as the major migrating motor complex passes through the obstructed segment. Waiting for the passage of currant jelly stool (bloody stool) before considering intussusception in the differential diagnosis is to be discouraged since this is a late finding. Early radiographic evaluation with plain films is to be encouraged. In fact, the possibility of intussusception should be considered when any type of blood in the stool is encountered.

In patients presenting similar to the above, but with lesser signs of obstruction, consideration should be given to vasculitis, far more commonly due to anaphylactoid (or Henoch-Schonlein) purpura than to Systemic Lupus Erythematosus. The typical presentation is dominated by crampy pain with a usually minor bleeding component. In the case of anaphylactoidpurpura, the platelet count may be high, but the ESR is typically in the normal range, in contrast to the elevation seen in SLE or to EBV-induced lymphoproliferative disease, both of which can mimic the radiologic picture of HSP. Treatment with corticosteroids is discouraged until these entities and lymphoma or leukemia are more definitively ruled out.

Inflammatory bowel disease (IBD) typically presents with a history of chronic but nonspecific signs. Poor weight gain and especially linear growth can be noted as much as 6 months before onset of cramping and bleeding, though there are hyper-acute variants of ulcerative colitis. These entities are covered in detail in a separate IBD chapter but for the purposes of this discussion, IBD can produce anything from occult bleeding to florid bloody diarrhea. In ulcerative colitis, the blood and stool texture are inversely related, with both mucusy diarrhea and bleeding being indicators of inflammation. Crohn’s disease, more commonly affecting portions of the GI tract other than the rectum, can present with unremarkable stools, but will also produce mucusy diarrhea if the distal colon is involved. A quick check of the oral mucosa and anus for the undermined vasculitic lesions seen in either site can be diagnostic of Crohn’s disease. A detailed family history searching for other versions of autoimmune disease can be supportive diagnostically, for while it is rare to have another with IBD in the family, there is often a strong family history of other autoimmune manifestations.

And finally, among the (relatively) common causes of colonic bleeding, polyps are to be considered whenever there is a report of painless bleeding of apparently moderate volume. Solitary juvenile polyps are the most common, and typically do not become large enough to cause bleeding before the end of the second year. As hamartomas, they are extremely vascular but have no sensory tissue and bear essentially no neoplastic risk as long as they are indeed solitary. The familial polyposis syndromes produce diffuse adenomatous polyposis, resulting in studding of the mucosa with often nearly confluent polyps, all of roughly the same size. These carry a significant neoplastic risk, and were until recently, an
indication for early colonic resection, but experience with NSAIDs (particularly sulindac) in adults has been extended to children in causing regression of the visible lesions. It remains to be seen if this significantly reduces the long-term neoplastic risk, but it seems to permit a reduction from the every-other-year colonoscopy surveillance often undertaken in the second decade. The diagnosis of polyps (single or multiple) starts with the history of painless bright red bleeding, generally without anemia despite a protracted history, and no anal fissure on inspection. Digital rectal examination is usually diagnostic as most solitary polyps arise within the last 2 inches of the rectum, and the familial adenomatous polyposis syndromes result in many small polyps within reach. Gardner's syndrome is associated with unusual retinal pigmentation in affected individuals and may also present with osteomas, though these usually are more prominent in the second decade while the small but visible bleeding usually appears by the middle of the first decade. Peutz-Jeghers syndrome is associated with pigmentation in unusual sites (buccal mucosa, the webbing between fingers and toes) but like the other multiple hamartomatous polyp syndromes, PJ typically does not present with bleeding. Therapy for isolated polyps is endoscopic removal and for multiple polyps is endoscopic sampling to establish a diagnosis. Waiting for a polyp to autoinfarct will not permit specific identification as to type, and the presence of more than 3 polyps, even with a "juvenile" type histology, is still associated with a higher risk of eventual colon cancer. Recent advances in genetic screening in the diagnosis and management planning of the familial adenomatous polyposis syndromes in pediatrics was recently discussed in detail in reference #6.

**Lower GI bleeding etiologies by age**

**Age** | Small volume | Medium volume | Large volume
---|---|---|---
Newborn | Infection, Ingested maternal necrotizing blood, sepsis, DIC enterocolitis | Infant Fissure, allergy, Same as infant plus volvulus DIC, peptic or other ulceration, infection, nodular intussusception, other bowel Meckel's diverticulum, CMV, lymphoid hyperplasia obstruction and/or ischemia MAI (esp. with HIV) | Child As above plus IBD As above plus HSP and All of above plus polyp(s), (Crohn's disease), cow's other vasculitides, typhilitis IBD (ulcerative colitis) milk protein sensitivity (immunosuppressed)
Infant | (with severe iron deficiency), | | (with severe iron deficiency),
| EBV (esp. immunosuppressed) | EBV (esp. immunosuppressed) | In summary, identification of "lower" GI bleeding is even more dependent on the history and characterization of the bleeding than that from the upper GI tract, and can be confounded by "upper" GI sources. Even with hemorrhage, patients rarely become significantly volume depleted on an acute basis and in most instances there is enough time to perform appropriate testing, including culture, in a sequential manner. Many times, the workup of the crampy patient with modest bleeding in loose mucusy stools involves a quick survey of inflammatory markers and a 2 to 3 day wait for the culture results from the rectal swab. A rectal swab has a superior yield over culture of stool material because the center of the lumen (i.e., stool material) typically contains dead or less viable organisms, while the viable enteric pathogens are closer to the mucosa and are more readily sampled by brushing the rectal wall. If the ESR is low in the face of thrombocytosis and a history of crampy pain, particularly in a patient with any dermal lesions, a small bowel series may demonstrate the characteristic "thumb-printing" of localized mucosal edema typical of HSP and enable early administration of corticosteroids. Otherwise, if the ESR is high and the growth parameters are low, once the culture results are found to be negative, the next step is colonoscopy to look for lesions indicative of IBD. On the other hand, finding a solitary polyp on initial examination permits a relaxed scheduling on a more elective basis both for the physician and the family.

For case #2, our patient's presentation with painless bleeding of apparent moderate volume yet without signs of significant volume depletion is indicative of a polyp. Rectal examination finds a single 1 cm pedunculated polyp 2 cm from the anal verge. Colonoscopy is scheduled, as the prep for even proctoscopy for polypectomy requires stringent removal of all stool to prevent a short-circuit current and an unintended burn, and a search is to be made for further polyps. The gastroenterologist reports no others are seen and the polyp in question has been easily removed by an electrocautery snare. Histologic analysis verifies a juvenile polyp, and no followup is planned.

**Questions**

1. You are called to the nursery where you are shown a burp cloth with loose clots of regurgitated blood. The newborn in question is sleeping quietly, with completely normal vital signs and no sign of tenderness or other bleeding when examined. You recall his mother presented with placenta previa. What do you do next?
2. At a two month well baby visit, his parents bring in a diaper double-bagged because of the foul odor. The stool is tarry and tests positive for occult blood, but the child appears particularly robust,
having gone from a birth weight of 7 pounds 1 ounce to his current weight of 12 pounds 10 ounces. He is somewhat fussy and demanding of feedings, and his mother complains of getting no rest as she has to feed him hourly. Recently, her left breast has become quite sore and there is intense pain when he nipples. On examination, the infant is colicky, but there is no abdominal tenderness and his vital signs are also within normal limits with no adjunct signs of intravascular volume depletion. What is going on?

3. A 3 year old presents with melena but no hematemesis, and no abdominal pain. How do you evaluate him?

4. Melena is usually indicative of upper GI bleeding. Indicate how this can sometimes be due to lower GI bleeding.

5. Red blood per rectum is usually indicative of lower GI bleeding. Indicate how this can sometimes be due to upper GI bleeding.

6. A 14 year old female has yet to show secondary sexual development which you have always attributed to excessive involvement with the school track team. However in the last 6 months her finishing times on the mile (her favorite event) have steadily lengthened from second best in the state to this week's race where she could not finish. She presents today complaining of loose stools, streaked with blood. How do you work up her illness?

7. A 3 year old boy presents to the emergency department passing bright red blood per anus. He is diaphoretic and tachycardic (120 supine, 140 upright) and complains of generalized abdominal pain. You are unable to localize tenderness but are comfortable that there is no rebound tenderness and he is not at risk of perforation. Placement of an NG tube to lavage his stomach is negative. By the time you have given enough crystalloid to replete his blood volume, his hemoglobin has dropped to 7 grams. Since his summer physical 2 months ago had included a hemoglobin of 12, you realize he has indeed lost a substantial portion of his blood volume over a short period of time. He is admitted to the hospital, where over the next two days as you wait for the stool culture results. He requires 250 cc transfusions daily to maintain his hemoglobin and you realize that the brisk bleeding continues. The stools remain bright red. What do you do next?

Answers to questions

1. A modified Apt test can be done. Take the loose clots and suspend them in a minimal amount of tap water (you need a visibly pink supernatant composed of free hemoglobin, hence the tap water to lyse the cells). Centrifuge the cells and add 5 cc of pink supernatant per 1 cc of 1% sodium hydroxide. Read in two minutes: adult hemoglobin turns yellow or brown, fetal hemoglobin remains pink. If the supernatant turns yellow, the blood is mother’s, and every one can relax.

2. This infant has no sign that the bleeding originates with him, as bleeding sufficient to produce melena should leave him quite shy. The history gives every sign that he has induced a mastitis (and nipple bleeding) in his mother, and she is able to compensate for the several ounces of blood loss that produced the melanotic stool. You counsel her on proper feeding and handling techniques to keep the infant satisfied without having to overfeed, and have his mother avoid feeding on the affected side until the inflammation subsides. At followup in a week, all are smiling.

3. ABC’s first. He shows no sign of acute intravascular volume depletion, but looks a little pale and turns out to be mildly anemic, indicating a longer standing problem. Next, place an NG tube to look for upper GI bleeding but you find no evidence of this. Now what? There is evidence of bleeding in an area bathed in acid, but it is not the stomach (or the duodenum). If he is hemodynamically stable, you have time to pretreat with a histamine-2 receptor blocker to improve the yield of a Meckel’s scan looking for ectopic gastric mucosa. This finds a hot spot in the lower mid-abdomen which the technician assures you is not tracer in the bladder. You contact your pediatric surgeon for minimally invasive removal of a presumptive Meckel’s diverticulum with acid-secreting ectopic gastric mucosa.

4. The black color is due to blood exposure to acid. Acid fermentation can take place in the cecum. If this occurs and the transit time is relatively slow, bleeding in this area can present as melena. Bleeding from a Meckel’s can also result in acid exposure in the lower GI tract.

5. The acid level in the stomach is low (possibly due to antacids and H2 blockers) and/or the bowel transit time is very rapid. Also, the bleeding may originate from the duodenum which does not expose the blood to acid if the pylorus is tight or the level of stomach acid is low.

6. The history has all the hallmarks of inflammatory bowel disease, but still the common things are more common. The physical examination shows no weight loss (but little net gain over the year), and she has a mild temperature elevation (100.5 degrees) and tachycardia (105) but no specific findings in the abdomen other than a mild increase in the amount of fluid and gas palpable in the small bowel and colon. Along with the CBC and ESR, you obtain a rectal swab for stool culture. There is no anemia, but the WBC count is slightly elevated and the ESR is 6. You are confused until the stool culture results return 2 days later, positive for Campylobacter. You call to discuss the
results and find her new puppy had been ill the week before (dogs can both harbor and become ill from this organism), and the poor race performance actually arose because she was getting fed up with her coach (her father) and had been wanting to quit. Since she is still out of school with the cramping and diarrhea, you start her on erythromycin, offer to act as a go-between on the issue of changing sports, and annotate her chart to remind yourself to monitor for other signs of depression in the future.

7. As the negative gastric aspirates over the last 2 days indicate no UGI source, you prep him for colonoscopy to look for a lower GI bleeding site. GoLYTELY is used in hopes of diluting the bleeding as blood rapidly absorbs all light even in a thin film, and you anticipate much suctioning and lavage which will markedly extend the time for the procedure. As he will be under anesthesia anyway, you also obtain consent for EGD for completeness' sake. At endoscopy, the EGD study finds the pylorus is tightly shut as there is a large duodenal ulcer (not a simple erosion) with a visible vessel (an indicator of high risk of recurrent bleeding). With this you joyfully cancel the colonoscopy as being unnecessary, and chalk up the experience as a reminder that rapid transit times and the low acid production of early childhood can sometimes prevent the blood from encountering enough acid to turn to acid hematin or melena. Indeed, the higher the volume lost, the more acid is needed and the less likely the reaction. Unfortunately, as the finding was a therapeutic surprise, you are unprepared to address the ulcer in any invasive manner (sclerotherapy, heater probe, etc.) and have to return the patient to intensive care on an IV histamine receptor blocker and carafate and sufficient antacid to keep the pH of the gastric contents, measured every hour, above 6.5 (and well above the 4.5 activation level of pepsin). Preparations are made to return with the proper equipment the next day if he continues bleeding, only to find the bleeding stops with the procedure (and the drop in splanchnic pressures encountered under anesthesia), the current measures are more than sufficient, and no further transfusions are required. The patient makes a rapid and full recovery, with no recurrence in over 5 years (based on actual personal experience).